Carbon Dioxide Embolism During Laparoscopy: Effect of Insufflation Pressure in Pigs

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ABSTRACT

Carbon dioxide embolism is a rare but potentially devastating complication of laparoscopy. To determine the effects of insufflation pressure on the mortality from carbon dioxide embolism, six swine had intravascular insufflation with carbon dioxide for 30 seconds using a Karl Storz insufflator at a flow rate of 35 mL/kg/min. The initial insufflation pressure was 15 mm Hg. Following recovery from the first embolism, intravascular insufflation using a pressure of 20 mm Hg at the same flow rate was performed in the surviving animals. Significantly less carbon dioxide (8.3 \pm 2.7 versus 16.7 \pm 3.9 mL/kg; p < 0.02) was insufflated intravascularly at 15 mm Hg than at 20 mm Hg pressure. All of the pigs insufflated at 15 mm Hg pressure with a flow rate of 35 mL/kg/min survived. In contrast, 4 of the 5 pigs insufflated at 20 mm Hg pressure died. The surviving pig died when insufflated with 25 mm Hg pressure following an embolism of 15.7 mL/kg. Intravascular injection was often associated with an initial rise in endtidal carbon dioxide tension, followed by a rapid fall in all cases where the embolism proved fatal. Insufflation should be begun with a low pressure and a slow flow rate to limit the volume of gas embolized in the event of inadvertent venous cannulation. Insufflation should immediately be stopped if a sudden change in end-tidal carbon dioxide tension occurs.

Key Words: Laparoscopy, Insufflation, Gas embolism, Carbon dioxide.

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INTRODUCTION

Carbon dioxide (CO₂) insufflation to achieve pneumoperitoneum during laparoscopic surgery has been utilized for over 25 years. It has emerged as the principle gas for insufflation because it is nonflammable and, in comparison with the other gases, is extremely soluble. Soluble gases such as CO₂ are much safer in the event of inadvertent gas embolism.¹ However, fatal gas embolism may still occur with CO₂ following unrecognized intravascular or intravisceral placement of the insufflating needle or trocar.²

During abdominal insufflation for laparoscopy, both the flow rate of CO_2 and the insufflation pressure can be varied. When the insufflation pressure is reached, the insufflator stops the flow of CO_2 . Most animal studies of gas embolism have investigated only the volume or type of gas embolized $\mathrm{gas}^{3,5}$ or, in some instances, the flow rate of embolized $\mathrm{gas}^{3,5}$ None of the studies has investigated the effect of the insufflation pressure on the mortality associated with carbon dioxide embolism, however.

We hypothesized lower insufflation pressures were safer in the event of inadvertent vascular injection of CO_2 because gas flow would cease as the pressure increased in the vein and limit the volume of gas embolized. The purpose of this study was to investigate the effects of insufflation pressure during CO_2 embolism in a pig model using a standard insufflation machine and a flow rate equivalent to what is commonly used in adult humans. Pigs were chosen because of the similarities of their cardiovascular and peripheral vascular systems to humans.⁸

MATERIAL AND METHODS

The study was done in six female swine $(28.7 \pm 6.2 \text{ kg})$ and was approved by the University of Minnesota Animal Care and Use Committee. Anesthesia was induced with pentobarbital (30 mg/kg iv); the swine were then endotracheally intubated with cuffed endotracheal tubes to ensure a tight laryngeal seal. The tidal volume was set at 20 mL/kg (FiO2 0.33) and respiratory rate $(14 \pm 4 \text{ breaths/min})$ adjusted to ensure a control PaCO₂ between 33 and 40 mm Hg. The animal was placed in

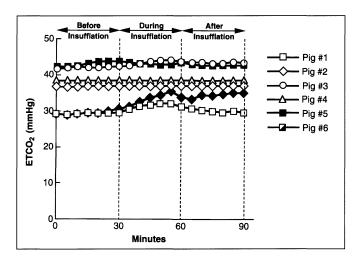


Figure 1. End-tidal carbon dioxide tension (ETCO₂) before abdominal insufflation, during insufflation and following insufflation prior to intravascular injection.

the supine position while being anesthetized with isoflurane 2%. End-tidal CO₂ tension was monitored continuously with a Nellcor N-1000/N-2500 (Nellcor Inc., CA) gas analyzer with airway gas sampling set at 150 mL/min (delay time 1755 ms; dynamic response of cuvette 17 ms, total response time 1932 ms). A pulmonary artery catheter was advanced into position via a right internal jugular cutdown. The femoral artery was cannulated by cutdown. The mean arterial pressure, pulmonary artery pressure, right atrial pressure, lead II of the ECG, and end-tidal CO₂ were monitored continuously and recorded on a computer at 10-second intervals using an automated data acquisition program. Arterial blood gases were checked before abdominal CO₂ insufflation, 10 minutes after insufflation and 10 minutes following deflation. More frequent (30 seconds and 10 minutes) arterial blood gases were obtained following intravenous CO2 insufflation.

Insufflation of CO₂

Initially CO_2 was inflated intraperitoneally for 30 minutes to observe the effects of abdominal insufflation on endtidal CO_2 , hemodynamics and survival. Intraperitoneal insufflation was performed using a trocar and a Karl Storz insufflator (Karl Storz Co., Culver City, CA). After this, the insufflated gas was removed. After a 30-minute recovery period, CO_2 was insufflated intravenously via a 16-gauge catheter inserted in an iliac vein cutdown.

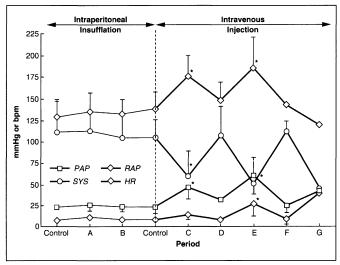


Figure 2. Systolic blood pressure (SYS), pulmonary artery pressure (PAP), right atrial pressure (RAP), and heart rate (HR) 10 minutes after abdominal insufflation (A), 10 minutes after deflation (B), 30 seconds after injection at 15 mm Hg (C), 10 minutes after injection (D), 30 seconds after injection at 20 mm Hg (E), 10 minutes after injection (F), and 30 seconds after injection at 25 mm Hg (G). *p < 0.05 compared to control by ANOVA.

Insufflation Pressures

The rate of insufflation was set at 35 mL/kg/min. This corresponded to the flow rate used for insufflation through a Veress needle by most surgeons in humans (2.4 liters/min for a 70 kg adult). During intraperitoneal insufflation, the maximum insufflation pressure was set at 15 mm Hg, and was maintained at this level during the 30-minute insufflation period. During intravenous insufflation, three consecutive insufflation pressures were studied: 15, 20, and 25 mm Hg. Each injection period lasted 30 seconds. By protocol, if the end-tidal CO2 tension decreased greater than 50 percent from the baseline value prior to the 30-second interval, a significant gas embolism was assumed to have occurred, and the insufflation was stopped to attempt to save the animal. A 30minute recovery period was allowed between injections. The volume of gas injected during each time period was recorded.

Reporting of Data

Graphic displays of the changes in end-tidal CO₂ during each insufflation along with accompanying alterations in

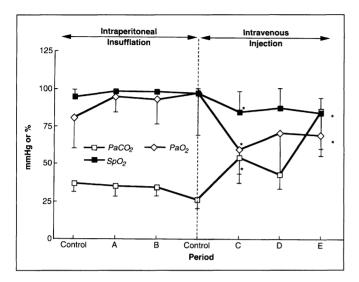


Figure 3. Arterial oxygen (PaO_2), arterial carbon dioxide tension ($PaCO_2$), and oxygen saturation (SpO_2) 10 minutes after abdominal insufflation (A), 10 minutes after deflation (B), 30 seconds after injection at 15 mm Hg (C), 10 minutes after injection (D), and 30 seconds after injection at 20 mm Hg (E). Arterial blood gases could not be obtained in the surviving pigs prior to and 30 seconds after the injection at 25 mm Hg. *p < 0.05 compared to control by ANOVA.

arterial blood gases and hemodynamics. The relationship between the intravenous insufflating pressure and the volume of CO_2 injected was determined. Data were reported as mean \pm SD. Significance (p < 0.05) was evaluated by ANOVA or Wilcoxon signed-rank tests.

RESULTS

Intraperitoneal injection of CO_2 resulted in only benign changes in end-tidal CO_2 , blood gases, and hemodynamics (**Figure 1-3**) with all animals surviving this phase. During iliac vein injection of CO_2 at 15 mm Hg pressure, one animal died immediately because the flow rate of CO_2 was accidentally set at 70 mL/kg/min (twice the standard rate). Because of the error, the data from this animal was excluded from further analysis. However, this animal was the only one in the series where the insufflation was stopped prior to 30 seconds because the end-tidal CO_2 fell below 50 percent of the initial value. Three of the surviving animals demonstrated an increase in CO_2 with the intravascular injection, while the other two demonstrated a marked drop followed by a gradual return to normal (**Figure 4**). The

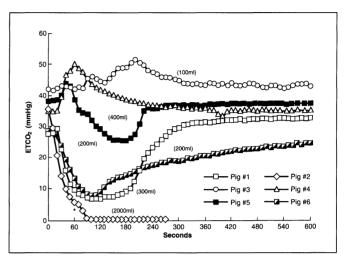


Figure 4. End-tidal carbon dioxide tension (ETCO₂) versus time for injection at 15 mm Hg pressure. *Injection was accidentally performed at twice the flow rate (70 mL/kg/min) called for by protocol (35 mL/kg/min).

changes in end-tidal CO2 were accompanied by a fall in the systemic blood pressure, an increase in the heart rate, and increases in pulmonary artery and right atrial pressures (Figure 2). The arterial CO₂ content increased, as expected, accompanied by a fall in the arterial oxygen tension and saturation (Figure 3). When the CO₂ injection pressure was increased to 20 mm Hg, four of the five animals demonstrated a drop in end-tidal CO2 followed by mortality within 10 min (Figure 5). Similar, but more profound changes in the hemodynamic and arterial blood gas values were noted after the second insufflation (Figure 2, 3). Death was immediately preceded by asystole or complete heart block in all cases. In the single surviving pig, the end-tidal CO2 increased followed by a return to a normal level. When the CO2 was injected at an insufflation pressure of 25 mm Hg, the single surviving pig demonstrated a transient rise in end-tidal CO₂ followed by a very rapid decline and mortality (Figure 6). At an insufflation pressure of 15 mm Hg, the volume of CO_2 injected was 8.3 \pm 2.7 mL/kg; this doubled to 16.7 \pm 3.9 mL/kg (p < 0.02) when the pressure was increased to 20 mm Hg. The volume injected in the surviving animal when the injection pressure was increased to 25 mm Hg was 15.7 mL/kg. When the injection volume was greater than 15 mL/kg all the pigs died.

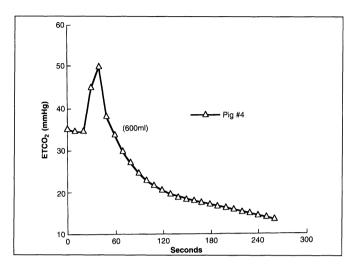


Figure 5. End-tidal carbon dioxide tension (ETCO₂) versus time for injection at 20 mm Hg pressure.

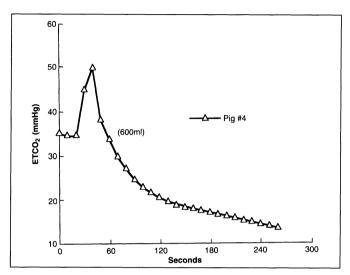


Figure 6. End-tidal carbon dioxide tension (ETCO₂) versus time for injection at 25 mm Hg pressure in the sole surviving pig.

DISCUSSION

Embolization of insufflating gas during induction of the pneumoperitoneum for laparoscopy is a sudden, dramatic event caused by accidental puncture of an intraabdominal vein or a vascular viscous. When enough gas is insufflated intravenously, it is rapidly carried to the vena cava and right atrium where it forms a gas lock, which in turn results in obstruction to venous return with a precipitous fall in cardiac output.9 Ventricular extra systoles or tachycardia, sinus bradycardia, complete heart block or asystole may result. Cardiac contractions break the gas up into small bubbles producing foam. When the foam reaches the pulmonary circulation, pulmonary hypertension and right heart strain results. Following CO2 embolization, an initial increase in end-tidal CO2 occurs reflecting CO2 excretion from CO2 absorbed into the blood. The abrupt drop in end-tidal CO₂ occurs as the pulmonary arterioles are blocked by the CO2 increasing alveolar dead space. Thus, a drop in end-tidal CO2 following insufflation means a significant, and potentially fatal, CO₂ embolism has occurred. 10-12

Fortunately, the incidence of ${\rm CO_2}$ embolism during laparoscopy is low (15 per 113,253 cases in gynecological laparoscopy).¹³ Careful surgical technique to avoid inadvertently cannulating or injuring a vein with the insufflating needle or trocar is the most important factor in preventing ${\rm CO_2}$ embolism in laparoscopy.² However,

the risk of ${\rm CO_2}$ embolism may be increasing as laparoscopic techniques are applied to more complex operations and patients with prior abdominal surgery.¹⁴

Studies have shown that the mortality from $\rm CO_2$ embolism is directly related to both the amount of $\rm CO_2$ injected and the rate of injection.^{1,3-7} Surgeons usually begin insufflation at a slow flow rate, then increase the flow as needed if insufflation is proceeding smoothly.

Our study suggests that surgeons should begin with a low insufflation pressure, as well. Most insufflation devices are designed to deliver the set flow rate until the intra-abdominal pressure begins to increase. For example, the Storz insufflator used in this study injects CO2 for 1.7 seconds then measures the pressure for 15 ms. The machine automatically slows down the flow rate as it senses pressure buildup in the abdomen, then eventually halts it at the set pressure. 15 Therefore, the pigs with the insufflation pressure set at 15 mm Hg received only one half of the volume of CO₂ in 30 seconds as when the pressure was set at 20 mm Hg in spite of having the same initial flow rate. The venous pressure could increase during intravascular injection of CO2 both from the local effects of the volume of gas itself and from right heart failure as a result of the embolism. At 20 mm Hg pressure or higher, the pigs received the amount of CO2 in 30 seconds predicted for a flow rate of 35 mL/kg/min (17.5 mL/kg), which most pigs could not survive.

In this study, 15 mm Hg was chosen as the lowest insufflation pressure tested because it is commonly used for laparoscopy in humans. Perhaps this pressure is too high. If the flow rate for CO_2 is set high and the patient has low venous pressure, significant and fatal CO_2 embolism could still occur at 15 mm Hg pressure. This was demonstrated in our study by the pig that accidentally was insufflated at twice the intended flow rate. Since the pressure in the iliac vein prior to insufflation in an adult human is, on average, 10 mm Hg, initial insufflation pressures lower than this value may limit or prevent altogether CO_2 embolism in the event of inadvertent venous cannulation. ¹⁶

Also, Wallace and Serpell, et al recently showed that patients undergoing laparoscopic cholecystectomies insufflated to 7.5 mm Hg pressure had less postoperative pain and better pulmonary function following surgery than those insufflated to 15 mm Hg.¹⁷ A similar study by Wakizaka and Sano, et al revealed that patients undergoing laparoscopic cholecystectomy had less hypercarbia if they were insufflated to 10 rather than 15 mm Hg.¹⁸ Lower insufflation pressures may, therefore, have other benefits in addition to minimizing the chance for CO₂ embolism.

Our study confirmed that a sudden decrease in end-tidal CO₂ was an early indicator of serious CO₂ embolism. Other devices have been suggested to detect gas embolism during laparoscopy, which are much more sensitive than end-tidal CO₂. For example, transesophageal echocardiography is very sensitive and can detect as little as 0.1 mL/kg of gas bubbles.¹⁹ Subclinical CO₂ emboli have been detected during laparoscopic cholecystectomies with this device.²⁰ However, transesophageal echocardiography is expensive, invasive, and readily available in many Transesophageal echocardiography may be too sensitive, picking up small emboli that have no importance. On the other hand, less invasive and sensitive Doppler devices, such as the transtracheal Doppler, may prove valuable.21

In summary, this experiment demonstrates that higher insufflation pressures result in a more severe ${\rm CO_2}$ embolism in the event of an inadvertent venous cannulation during insufflation. Surgeons should keep both the insufflation pressure and flow rate low until they are certain uneventful abdominal insufflation is occurring. Insufflation should be immediately halted if there is a decline in the end-tidal ${\rm CO_2}$.

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